DISSERTATION ON

THE IMPACT OF MANAGEMENT OF CHRONIC RHINOSINUSITIS IN THE CLINICAL COURSE OF BRONCHIAL ASTHMA.

SUBMITTED FOR

M.S.DEGREE EXAMINATION

BRANCH IV OTORHINOLARYNGOLOGY

STANLEY MEDICAL COLLEGE

THE TAMILNADU

DR.MGR MEDICAL UNIVERSITY

CHENNAI

MARCH 2007.

CERTIFICATE

This is to certify that the Dissertation titled **THE IMPACT OF MANAGEMENT OF CHRONIC RHINOSINUSITIS IN THE CLINICAL COURSE OF BRONCHIAL ASTHMA** is the bonafide research work done by **DR. V.V. RAMACHANDRAN** under my guidance, supervision and to my satisfaction during the period 2004 – 2007 and submitted to the **TAMILNADU DR MGR MEDICAL UNIVERSITY** towards partial fulfillment for the degree of **MASTER OF SURGERY** in the subject **OTORHINOLARYNGOLOGY**.

PROF. JACINTH CHELLAIAH

PROFESSOR & H.O.D

DEPARTMENT OF OTOLARYNGOLOGY,

STANLEYMEDICALCOLLEGE,CHENNAI

ACKNOWLEDGEMENT

I am thankful to **Prof. D.R.GUNASEKARAN,DEAN,**Government Stanley Medical College & Hospital for granting me permission to do the Dissertation work and utilising hospital facilities.

I am extremely grateful to **Prof.JACINTH CHELLAIAH**, Professor and H.O.D,Department of Otorhinolaryngology,Stanley Medical College, for his exemplary guidance and endless support through out this endeavour.

I genuflect before **Prof.A.MURALEEDHARAN**,Additional Professor ,Department of Otorhinolaryngology,Stanley Medical College, holding whose finger I can walk through all the way of darkness as a child due to the Care,Concern and Confidence he exercised on me.

I am thankful to **Dr.M.N.SHANKAR, DR.P.ILANGOVAN, DR.M.GOWRISHANKAR and DR.S.UDAYASHANKARAN**, Assistant Professors,Department of Otorhinolaryngology Stanley Medical College.

I am immensely grateful to **DR. R. THALAPATHI RAMKUMAR** for his enduring encouragement and enthusiastic support.

I am specially thankful to Prof. **S. SHIVAKUMAR,** Professor of Therapeutics Department of Medicine for his guidance.

I am indebted to thank **DR.N KARUNAGARAN,** Asst.Prof.,Department of Medicine & **DR. D. RAVISHANKAR M.D**.(General medicine) Post graduate for helping me in screening the Bronchial asthma cases.

I am thankful to **DR.SENTHIL** Lecturer, Community Medicine, who guided me in the Statistical works. I thank **DR.P. KUMAR** & other Junior Post Graduates for their endless support.

I express my gratitude to my sister **Mrs..IDAYAKANI VENKATAKRISHNAN** & aunt **Mrs.VIJAYALAKSHMI SRINIVASAN** for their painstaking help in preparing this project.

I am also grateful to my patients as they are the centre around which the planets of medicine revolve round.

At last I would like to dedicate this work to the living god,my mother, **Mrs.SURIYAVEDHACHALAM** and my father **Mr. PULAVAR VEDHA,** who is blessing me from heaven.

CONTENTS

PREFACE

Bronchial asthma and rhinosinusitis are common respiratory diseases that often coexist . Furthermore and of considerable interest is the possiblity that severity of asthma is influenced by the upper airway diseases. The influence and interaction between asthma and rhinitis exist with chronic rhinosinusitis, allergic rhinitis and viral respiratory tract infection. These observations raise the possiblity that events in the upper respiratory tract can influence lower respiratory tract. The first suggestion of possible association of upper and lower respiratory tract pathology comes from the studies demonstrating that asthmatics have abnormal paranasal sinus radiographs(1).

The next logical step performed was to see if aggressive treatment of paranasal sinus diseases could improve asthma . Many symptoms and signs are shared between upper and lower respiratory tracts and its important that these symptoms should consider differential diagnosis of both systems . Although historically it was believed that structurally and functionally there are differences within the respiratory tract which is used to divide it in to the upper and lower respiratory tracts . Its now being appreciated the concept of one airway and one disease. Despite many studies supporting an association between rhinosinusitis and bronchial hyper responsiveness, controversy still remains whether treating one airway disease can ameliorate another?(7).

AIM

To study the impact of the management of Chronic rhinosinusitis in the clinical course of Bronchial asthma in terms of patients symptoms subjectively & Peak expiratory flow rate objectively.

HISTORY

During 2nd Century A.D.,Galen observed relationship between nasal infection and wheezing.

In 1919 , Sluder hypothesised Sinopulmonary reflex. (12).

In 1928, French physiologist Kratchmer observed stimulation of nasal mucosa and bronchial hyperresponsiveness.

In 1969, Kaufman and Wright found silica particles applied to nasal mucosa increase lower airway resistance(3).

ANATOMY OF THE AIRWAY

EMBRYOLOGY OF NOSE AND NASAL CAVITY:

At about 4 weeks of intrauterine life bilateral ectodermal thickenings appear in the frontonasal process above the stomatodeum called as nasal placode (or) olfactory placodes.

The nasal placodes invaginates to form the olfactory (or) nasal pit & it is continous with the stomatodeum below. Proliferating mesoderm around the pit raises above the surface medially & laterally to from the medial & lateral nasal process respectively .

The nasal pits deepens to form nasal sac by 5 wks.Nasal sacs expand dorsally & caudally,separated from stomatodeum by bucconasal membrane or oronasal membrane with progressive thinning of mesoderm dorsally & caudally . The maxillary process of the 1st arch fuse with medial nasal fold & frontonasal fold which closes the nasal pit in to widely separated primitive nasal cavities.

The bucconasal membrane separating primitive nasal cavity & stomatodeum breaks at 14-15mm stage. Primitive posterior nasal aperture is in horizontal plane.

The maxillary process fuses with lateral nasal fold forming the nasomaxillary groove which later canalises to form nasolacrimal duct.

Lateral nasal fold forms nasal bone, upper lateral cartilage & lower lateral cartilage.

Medial nasal fold forms Premaxilla , upper lip & medial crus of lower lateral cartilage.

Mesoderm of the lateral part of the upperlip is derived from maxillaryprocessand philtrum from the frontonasal process.

Primitive palate begins to form anteriorly by fusion of maxillary &frontonasal processes.

A midline ridge develops from the posterior edge of the frontonasal process in the roof of stomatodeum to the rathke's pouch which forms nasal septum.

As the nasal cavity enlarges the palatal process of maxillary mesoderm grows medially towards each other & the septum. Fusion occur from anterior to posterior.

DEVELOPMENT OF TURBINATES:

Elevations in lateral wall appear by about 9 – 10 weeks as major furrows. It has a ascending & desending ramus.

Maxillo turbinal becomes inferior turbinate

Ascending ramus of 1st ethmo turbinal becomes agger nasi

Desending ramus 1st ethmoturbinal becomes uncinate.

Ascending ramus of 1st primary furrow becomes frontal recess.

Desending ramus of 1st primary furrow becomes Hiatus semilunaris and Ethmoidal infundibulum.

2nd ethmoturbinal becomes middle turbinate

3rd ethmoturbinal becomes superior turbinate .

4th & 5th ethmoturbinal fuses to form supreme turbinate .

Turbinates are really only the ends of bony lamella which traverse the entire ethmoid laterally to lamina papyracea & superiorly to lamina cribrosa.

1st lamella - uncinate

2nd lamella – bulla lamella

3rd lamella – ground lamella

4th lamella – superior turbinate

5th lamella – supreme turbinate.

DEVELOPMENT OF LUNGS :

Each lung develops from a bud at the lower end of laryngotracheal tube that grows down from the floor of the primitive pharynx. This epithelial buds form only the epithelial part of the lung; the connective tissue, cartilage and muscle of the bronchial tree are derived from then surrounding mesoderm. By the 5^{th} month the lung has a glandular appearance, with clumps of epithelial cells and hardly any recognizable lumina,but the cell group become expanded and lie adjacent to a multitude of capillaries so that by the 7^{th} month there are sufficient alveoli to sustain a viable infant following premature birth at this time. Surfactant begins to be secreted about the 6^{th} month.

THE LATERAL NASAL WALL:

The inferior meatus is that part of the lateral wall of the nose lateral to the inferior turbinate.It is the largest meatus, extending almost the entire length of the nasal cavity. The meatus is highest at the junction of the anterior and middle third.

In adults this ranges from 1.6 to 2.3cm at 1.6cm along the bony lateral wall . The nasolacrimal duct opens into the inferior meatus usually just anterior to its highest point. There is no true valve, the opening being covered by small folds of mucosa. It can be identified in life by gentle massage of the lacrimal sac at the medial canthus.

Inferior turbinate

This structure is composed of a separate bone, the inferior concha which has an irregular surface, perforated and grooved by vascular channels to which the mucoperiosteum is firmly attached.

The bone has a maxillary process which articulates with the inferior margin of the maxillary hiatus. It also articulates with the ethmoid ,palatine and lacrimal bones completing the medial wall of the nasolacrimal duct. The inferior concha has its own ossification centre which appears around the fifth intrauterine month .

The turbinates possesses an impressive submucosal cavernous plexus with large sinusoids under autonomic control which provides the major contribution to nasal resistance. The turbinate is covered by respiratory epithelium with a high number of goblet cells which decrease in density towards the posterior end.

Middle meatus

The middle meatus is that portion of the lateral nasal wall lying lateral to the middle turbinates. It receives drainage from the frontal, maxillary and anterior ethmoidal sinuses.

Considerable confusion has arisen with regard to terminology in this area, as many term orginally defined in the last century by the german and french have been used interchangeably. In the past when radical sinus surgery was predominantly used for most pathology this was of less significance. The advent of endoscopic surgery has led to an increased interest in the detailed anatomy of the region and a need for consensus in terminology.

The configuration of the structures of the middle meatus are complex and variable, but can more readly be understood when the embryological development of the area is considered. If the topographical anatomy is considered in the sagittal plane, a number of structures are apparent, covered by the middle turbinate in a disarticulated skull the maxillary bone has large opening in its medial wall the maxillary hiatus.

In the articulated skull this is filled in by adjacent bones.
Inferior: the maxillary process of the inferior turbinate bone.
Posterior: the perpendicular plate of the palatine bone
Anterosuperior: a small portion of the lacrimal bone.
Superior: the uncinate process and bulla of the ethmoid.

A portion of the maxillary hiatus is nevertheless left open by these osseous attachments, which in life is filled by the mucous membrane of the middle meatus, the mucous membrane of the maxillary sinus and the intervening connective tissue-the membranous portion of the lateral wall. This membranous area can be defined as lying anterior or

posterior to the uncinate process ,constituting the anterior and posterior fontanelles respectively.

It is in the fontanelles that accessory ostia are found, their formation probably arising as a consequence of infection and consequently, they have been compared to perforations in the tympanic membrane.

It is difficult to ascertain a natural incidence for accessory ostia but is probably of the order 4-5% in the general adult population inceasing to 25% in patients with chronic rhinosinusitis. Accessory ostia are found most frequently iin the posterior fontancelle which is generally larger than its anterior counterpart.

The uncinate process

This thin crescent of bone curves posteriorly, parallel with the curve of the anterior face of the ethmodial bulla. In addition to its anterior attachment to the maxillary hiatus it attaches superiorly in a variety of ways.

It may curve laterally to reach the laminapapyracea. It may attach superiorly to the skull base or occasionally it may fuse with the insertion of the middle turbinate. In the two later situations there is confluence of drainage from the frontal and maxillary sinuses

with the ethmoidal infundibulum leading into the frontal recess with obvious potential pathological consequences.

When the uncinate process inserts on the lamina papyracea, the ethmoidal infundibulum leads superiorly into a blind pouch, **the terminal recess** . Exceptionally, the unicinate process is pneumatized.

The agger nasi

This area has caused considerable confusion but constitutes with the uncinate precess, the remnants of the nasoturbinal in lower mammals and is the most anterior part of the ethmoid. It is represented by a small crest or mound on the lateral wall just anterior to the attachment of the middle turbinate .

It is occasionally pneumatized, though the incidence in the normal population is probably less than 5%. Pneumatization of the agger nasi region may encroach upon the nasolacrimal duct.

Hiatus semilunaris

The hiatus semilunaris is a two dimensional space lying between the posterior edge of the unicate process and the anterior surface of the ethmoidal bulla. The ethmoidal infundibulum is reached from the middle meatus by passing through the hiatus semilunaris.

Ethmodial infundibulum

The ethmodial infundibulum is a three- dimensional funnel connecting the natural ositum of the maxillary sinus to the middle meatus via the hiatus semilunaris. It is defined:

Medial:uncinate process and hiatus semilunaris

Lateral:lamina papyracea

Anterior:an acute-angled blind recess where the uncinate process meets the lamina papyracea.

Posterior:anterior face of the ethmoidal bulla

Superior:this will vary according to the attachment of the uncinate process.

Anteriorly the uncinate process may be separtated by only 1-2 mm from the lamina papyracea for some distance before they join.

The natural ostium of the maxillary sinus lies in the floor of the ethmodial infundibulum usually at th junction of its middle and posterior third and so is not readily visualized until the unicinate process has been removed.

The frontal recess

The frontal recess is found in the most anterosuperior portion of the middle meatus.The term frontonasal duct has been generally abandoned as no true duct exists either histologically or topographically in most people. The natural ostitum of the frontal sinus is somewhat variable in its configuration but most frequently it presents as an hour-glass

narrowing opening directly into the recess. Rarely a longer narrowed region is found.in 10 % of patients ,mutiple ostia are found , though these openings should not be confused with more laterally placed supra bullar cell running in to orbital roof.

BOUNDARIES OF FRONTAL RECESS

- Laterally , lamina papyracea.
- Medially , middle turbinate.
- Superiorly, skull base.
- Inferiorly and anteriorly, roof and posterior wall of ethmoidal bulla.
- Posteriorly , basal lamella of middle turbinate.

The ethmoidal bulla

This is one of the most constant features in the middle meatus containing the largest anterior ethmoidal cell .

It may be poorly aerated or completely unpneumatised in 8 % of patients and it is called as **Torus lateralis.**

The anterior face forms posterior margin of the hiatus semi lunaris and ethmoidal infundibulum. Posteriorly the bulla may fuse with the basal lamella of the middle turbinate and superiorly it may reach the roof of ethmoid forming the posterior wall of frontal recess.

Sometimes a cleft is encountered between the posterior wall of bulla and the basal lamella of the middle turbinate , the lateral sinus or recessus supra ethmoidalis. If the bulla does't reach the skull base lateral recess will connect above the bulla with frontal recess anteriorly.

Anatomical variations

There is a considerable range of anatomical variation in this area which has been implicated in the aetiology of sinus infection.This includes pneumatization of the middle turbinate , enlargement of the ethmoidal bulla, a paradoxically bent middle turbinate, everted uncinate process and the presence of haller cells or a septal deflection.

The incidence with which these are seen in a normal population may appear to be less frequent than in those individuals with chronic rhinosinusitis but no closer inspection it is clear that it is narrowing of the ostiomeatal complex rather than the existence of the variant which is the important factor.

Superior meatus

This meatus is again defined by its relationship to the superior turbinate . The Posterior Ethmoidal cell open into the region. A Supreme turbinate is discernible above the superior meatus in 60-67% of subjects,though is well developed in less than 20%.

Drainage to the corresponding supreme meatus from the posterior ethmoidal system can take place under these circumstances.

Sphenoethmoidal recess

The sphenoethmoidal recess lies medial to the superior turbinate and is the location of the ostium of the sphenoid sinus.

Histology of the lateral wall :

The majority of the lateral wall is covered by respiratory ciliated columnar epithelium though there is a small variable area superiorly of olfactory epithelium spreading down from the cribriform plate. Areas of squamous metaplasia are often found on the lateral wall, particularly in areas subjected to greatest airflow such as the anterior end of inferior turbinates.

ARTERIAL SUPPLY :

- Sphenopalantine artery
- Greater palantine artery
- Anterior ethmoidal artery
- Posterior ethmoidal artery

VENOUS DRAINAGE:

- Ethmoidal vein
- Facial vein
- Sphenopalatine vein

NERVE SUPPLY :

- Anterior ethmoidal nerve
- Branches of sphenopalantine ganglion
- Greater palantine nerve
- Anterior superior alveolar nerve.

NASAL EPITHELIUM :

Within the epithelium three types of cells are identified

- Basal cells lying on the basement membrane
- Ciliated columnar cells
- Goblet cells

NASAL SUBMUCOSA:

Nasal submucosa lies beneath basement membrane and contains a host of cellular components in addition to nasal glands, nerves and blood vessels. In light microscopy of nasal biopsy the predominant cell in the submucosa was mononuclear cells. Three types of nasal glands are present,

1. anterior serous glands
2. seromucous glands
3. intraepithelial glands.

BRONCHOPULMONARY SEGMENTS

a. Right upper lobe	**a. Left upper lobe**
1. Apical segment	1.Apicoposterior segment
2. Posterior segment	2.Anterior segment
3. Anterior segment	
b. Right middle lobe	**b. Lingula**
1. Lateral segment	1.Superior segment
2. Medial segment	2.Inferior segment
c. Right lower lobe	**c.Left lower lobe**
1. Apical segment	1.Apical segment
2. Medial basal segment	2.Medial basal segment
3. Anterior basal segment	3.Anterior basal segment
4. Lateral basal segment	4.Lateral basal segment
5. Posterior basal segment	5. Posterior basal segment

Structure of Broncho Pulmonary Segment:

Bronchi have smooth muscle and hyaline cartilage in their walls and are lined by the typical respiratory type of epithelium pseudostratified ciliated columnar with mucous glands. By successive divisions they become smaller and smaller, when cartilage disappears(at a diameter of about 1 mm) bronchi become bronchioles. The more distal divisions of bronchioles, where cilia begin to disappear, are respiratory bronchioles, so called because some alveoli(air sacs) open off them. The bronchioles just proximal to respiratory bronchioles are rather confusingly called terminal bronchioles.Beyond the respiratory ducts with many alveoli opening off them,and these continue in to alveolar ducts.

Blood supply :

The bronchial tree receives its arterial supply from 3 bronchial arteries, on left side as direct branches of aorta and on right side from posterior intercostal arteries. The bronchial veins drain in to azygos vein on the right side and hemiazygos vein on left side.Pulmonary artery divides with the bronchus but does not supplies it. It supplies the alveoli. Pulmonary veins does not follow the bronchi but runs in the intersegmental septa.

PHYSIOLOGY OF AIRWAY

PHYSIOLOGIC FUNCTIONS OF THE NOSE:

1.Respiration

- Heat exchange
- Humidification
- Filtration
- Nasal resistance
- Nasal fluids & ciliary function
- Neurovascular reflexes
- Voice modification

2.Olfaction .

Nose modifies the air so that gas exchange in alveoli can be achieved without damaging it. During inspiration the air flow is opposite to the direction of blood flow, thus it is more efficient in warming inspired air. Inspired air is warmed from a temperature of around 20^{o} c to 31 ^{o}c in the pharynx and 35 ^{o}c in the trachea.

As the expiratory air passes through the nose, it gives up heat to the cooler nasal mucosa and the temperature falls so that water condenses on it.

During inspiration the airflow is directed upwards and back wards mainly over the anterior end of the inferior turbinate. It divides it in to two above and below the middle turbinate rejoining in the choanae. Its considered to be laminar, though practically its turbulent in the olfactory region. During Expiration ,air flow is turbulent with eddy currents.

NASAL RESISTANCE

Nose accounts for half of the total airway resistance. The resistance is important during expiration since the positive pressure is transmitted to the alveoli & keeps the lung expanded. The air flow & nasal resistance are modified by mucosal changes during nasal cycle. Cyclical changes take place every 4 – 12hrs. They are constant for each person and nasal cycle is controlled by autonomic nervous system.

PROTECTION OF LOWER AIRWAY

Mechanical & chemical protection of the lower airway is one of the major functions of the nose. Nose is able to remove particles of 30microns(or) more from air.

NASAL SECRETIONS

- Water & ions from transudation.
- Glycoprotein: Sialomucins, fucomucins,Sulfomucins.
- Enzymes: Lysozymes,Lactoferrin.
- Immunoglobulin: 1gA,IgE, IgG, IgM, IgD.
- Circulatory proteins:Complement, alpha2 macroglobulin

Lactoferrin acts by binding to divalent metal ions & prevents the growth of certain bacteria particularly staphylococcus and pseudomonas.

Principles of secretion transport:

Drainage and ventilation are two important functions in the maintence of normal physiology of paranasal sinuses and their mucous membrane . Normal drainage of the Paranasal Sinuses is a complex function of both the secretion and transport mechanisms,and to a larger extent dependent on the amount of mucus produced,its composition ,effectiveness of ciliary beat and condition of the ostia and the ethmoidal clefts in to which respective sinus ostia opens.

In health the mucous blanket that covers the nasal mucosa is continuosly produced by sreomucinous glands & intraepithelial goblet cells.This mucus film has 2 layers:an inner serous layer, called **sol phase,** in which cilia beat and outer more viscous layer **gel phase.**

Normal nasal mucous exists in equilibrium between the sol layer and gel layer at a p^H of 7.5-7.6.A proper balance between the inner sol phase and outer gel phase is of critical importance for normal mucociliary clearance.

Dust particles are incorporated in to the gel phase and toghether with the gel phase transport out of the sinuses in normal conditions.Under abnormal conditions pathogens may be incorporated in to cells of mucosa.

An unimpeded flow of air during inspiration through the nose is further a factor since forced inspiration creates suction or negative pressure that promotes transportation of mucous out of the sinuses.

The cilia beat in a synchronised(transversally) and metachronised(longidudinally) manner.The cilia move almost exclusively in the sol phase of secretion.The gel phase is actively transported over the sol phase like a carpet during their active cilia beat; there is no contact between the cilia and the gel phase during the recovery phase.

Under normal conditions, the mucous layer is steadily transported away.Endoscopic studies indicate that a healthy maxillary sinus renew its mucus layer every 20 to 30 minutes.

Bony crests,found protruding in to the lumen of the frontal sinuses are usually traversed by the secretions with out any problem.If the secretions becomes more viscous and consequently thicker and heavier,however ,these crests may become a obstacle to mucus transport and the secretion may be retained at the crest and finally drain away through the effect of gravity.

Human nasal and paranasal sinus mucosa and their ciliary activity may survive the death of individual for 24 to 48 hours.With progressing dehydration and increasing viscosity of the mucous or with the final death of mucosal cells, the ciliary beat ceases.

One of the most important of the Messerklinger' s discoveries following earlier animal studies by Hilding was the observation that the secretions of various sinuses do not raech the respective ostia by random fashion,but follow very definite pathways,which seems to be genetically determined.

Secretion transport in the maxillary sinus:

In the maxillary sinus secretion starts from the secretion starts in the floor of the maxillary sinus in a stellate pattern. The mucuos is transported along the anterior ,medial , posterior, and the lateral walls of sinuses as well as along the roof.All the secretions converge at the natural ostium of the maxillary sinus.

The maxillary sinus ostium normally opens in to the floor of posterior third of ethmoidal infundibulum.Secretion from the sinus is always transported via the natural ostium even when there is one or more accessory ostium in the area of the fontanelles and even in the patients in whom a patent window in the inferior meatus is created.

While the inferior meatal antrostomy can provide ventilation to the diseased sinuses and consequently may help normalise the sinus, these inferior meatal antrostomy do not achieve considerable active outwardly directed transport of secretions.

Secretion transport in frontal sinus:

In the frontal sinus there is an active inwardly directed transport of mucous.along the interfrontal septum,mucus is transported in to the frontal sinus,then laterally along the roof and back medially via the floor and the inferior portion of the posterior and the anterior wall of the sinus.The secretion then exits the frontal sinus after one round trip.This is the result of a whorl- like motion in the ciliary pattern.

An isolated mucosal lesion as long as it does not obstruct the ostium usually does not obstruct mucus transport.If however the mucosal surfaces come in direct contact with each other, a **Bridging Phenomenon** may occur, with more serous component of the mucus remaining in the recess.Even though the drowned cilia beat in vain ,the gel phase is transported because of its cohesion.

NASAL IMMUNE SYSTEM

1. Surface properties

2. Mechanical

Physical characteristics of mucus

3.Innate immunity

a) Bactericidal activity of mucus

b) Proteins: Lactoferrin, lysozymes, Macroglobulin.

c) C reactive Protein, Complement system.

d) Cellular: Polymorphs and macrophages.

4.Acquired immunity

a) Surface 1gA, IgM, IgE &IgG

b) Lymphocytes – Mucosal associated lymphoid tissue

5.Distant sites

Adenoids & lymph nodes

NASOPULMONARY REFLEX:

Increased air flow through one side of the nose is associated with increased ventilation of homolateral lung. Blowing of air through a nostril causes bronchial muscle to relax on the same side and increase its respiratory activity.

THE POSSIBLE FUNCTIONS OF PARANASAL SINUSES:

1.Vocal resonance and diminution of auditory feedback .

2.Air conditioning.

3.Pressure damper.

4.Reduction of skull weight.

5.Mechanical rigidity.

6.Heat insulation.

7.Increasing the olfactory area.

PULMONARY VOLUMES(11).

Tidal volume is the volume of air inspired (or) expired with each normal breath. It amounts to about 500ml in the adult male.

The inspiratory reserve volume is the extra volume of air that can be inspired over and above the normal tidal volume, when the person inspires with fullforce and it is usually equal to about 3000ml.

The expiratory reserve volume is maximum extra volume of air that can be expired by forceful expiration, normally about 1100ml.

The residual volume is the volume of air remaining in the lungs after the most forceful expiration and it averages about 1200ml.

PULMONARY CAPACITIES

The inspiratory capacity equals the tidal volume plus inspiratory reserve volume . This is the amount of air a person can breathe in beginning at the normal expiratory level and distending the lungs to maximum amount. It averages about 3500ml.

The functional residual capactiy equals the expiratory reserve volume plus the residual volume. This is the amount of air that remains in the lungs at the end of normal expiration and it is about 2300ml.

The vital capacity = Inspiratory Reserve vol.+Residual vol. +expiratory reserve vol.

This is the maximum amount of air a person can expel from the lungs after filling the lungs to their maximum extent in then expiring to maximum extent.

The total lung capacity is the maximum volume to which the lungs can be expanded with the greatest possible effort (5800ml) it is equal to the vital capacity + residual volume.

Minute Respiratory volume = Respiratory rate x Tidal Volume.

Minute Respiratory volume = 12 x 500ml = 6000ml/min.

ALVEOLAR VENTILATION:

Alveolar ventilation is the rate at which new air reaches the alveoli, alveolar sacs, alveolar ducts and respiratory bronchioles.

Alveolar ventilation = Respiratory rate x (tidal volume –dead space air)

= 12 x (500 –150ml) =4200ml/min.

DEAD SPACE AIR :

Some of the air a person breathes never reaches the gas exchange areas but fills respiratory passages where gas exchange doesn't occur such as the nose , Pharynx & trachea.This air is called dead space air because it is not useful for gas exchange and the normal dead space air is about 150ml.

To keep the trachea from collapsing ,multiple cartilage rings extend above 5/6 of the wayaround the trachea. In the walls of the bronchi, less extensive curved cartilage plates alsomaintain a reasonable amount of rigidity yet allow sufficient motion for the lungs to expand. The bronchioles are not prevented from collapsing by rigidity of their wall instead they are kept expanded by transpulmonary pressure that expand the alveoli.

In all areas of the trachea & bronchi not occupied by cartilage plates the walls are composed mainly of smooth muscle. Also the walls of the bronchioles are almost entirely smooth muscle, with the exception of the most terminal bronchiole called respiratory bronchiole which is mainly composed of pulmonary epithelium & under lying fibrous tissue.

Under normal respiratory conditions air flow through the respiratory passage ways so easily that less than 1cm of water pressure gradient from the alveoli to the atmosphere is sufficient to cause enough airflow for quite breathing. The greatest amount of resistance to airflow occur not in the minute air passage of terminal bronchiole but in some large bronchioles, bronchi & near trachea.

Yet in disease condition the smaller bronchioles often play a for greater role in determining airflow resistance because of their small size.

1.Muscle contration in their walls.

2.Edema occuring in the walls.

3 Mucus collecting in the lumen of the bronchioles & Parasympathetic constriction of the bronchioles .

PATHOPHYSIOLOGIC MECHANISMS

WHO definition of Bronichal asthma(8):

Bronchial asthma is a chronic inflammatory disorder of the airways in which cellular elements play a role, in particular mast cells ,eosinophils, T lymphocytes macrophages, neutrophils and epithelial cells. In susceptible individuals this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness and coughing .These sysmptoms usually associated with widespread but variable airway obstruction that is often reversible either spontaneously (or) with treatment. The inflammation also causes an associated increase in existing bronchial hyper responsiveness to a variety of stimuli.

SYMPTOMS OF ASTHMA

- Wheeze
- Chest tightness
- Cough
- Breathlessness.

TYPES OF BRONCHIAL ASTHMA:

Extrinsic- in which identifiable external trigger factor are present.

Intrinsic- in which no such factors are identified.

OVERVIEW OF PATHOLOGICAL CHANGES IN ASTHMA:

- Edema of the airway wall .
- Shedding of the airway epithelium.
- Mucus plugging of the airway lumen
- Thickening of the reticular layer of basement membrane.
- Goblet cell hyperplasia
- Neovascularisation
- Vasodilatation in the smooth muscle layer .
- Hyperplasia &hypertrophyof airway smooth muscle.
- Mucosal gland hypertrophy.
- Ultimately the development of irreversible fibrosis.

CURRENT VIEW OF MECHANISMS IN ASTHMA.

Asthma is now viewed as a complex inflammatory condition involving many inflammatory cells which release a wide variety of mediators. These mediators act on cells of the airway leading to potentially reversible abnormalities including smooth muscle contraction ,mucus hypersecretion,plasmaleakage,edema,activation of cholinergic reflexes and activation of sensory nerves.

Chronic inflammation leads to structural changes including goblet cell hyperplasia,new vessel formation ,subepithelial fibrosis, smooth muscle hypertrophy and hyperplasia

SINUS & BRONCHIAL SIMILARITIES

1. Atmospheric exposure
2. Ciliary epithelium
3. Goblet cells producing mucus
4. Aerosols (Infection (or)allergens) trigger pathogenic events
5. Defence mechanism.
6. Eosinophilic inflammation.

SINO PULMONARY DISORDERS

1. Sino bronchial allergy
2. Sinopulmonary syndrome
    - Sinobronchial allergic mycosis
    - Primary ciliary dyskinesia
    - Wegener granulomatosis
    - Churg strauss syndrome.
3. Cystic fibrosis

The frontal and the maxillary sinuses are dependant sinuses, subordinate to the their prechambers in the ethmoid and lateral nasal wall.Drainage and ventilation are essential for the normal funtioning of the sinuses.The ventilation and drainage of the maxillary and the frontal sinuses pass through very narrow and complicated clefts before they reach the free middle meatus.

Those disorders which produce any additional stenosis of these narrow key areas resulting in the contact areas of the opposing mucosa with mucuos retention .If infected smaller or larger areas of chronically diseased mucosa may persist.Frequently these areas of intimate contact can be identified as the site of origin of polyps.

Messerklinger noted that limited resection of disease in the key areas and reestablishment of drainage and ventilation via natural pathways,even massive mucosal pathology usually healed without direct inervention in these sinuses.

Chronic Rhinosinusitis is defined as inflammation of the mucosal lining of nose and sinuses lasting for atleast 12 weeks duration(31).

CELLULAR PATHWAY:

Chronic sinusitis and asthma are characterised by an inflammatory process that is marked histologically by eosinophils.The eosinophils contains major basic protien as well as eosinophilic cationic protien both of which contribute to inflammation and injury of epithelium of nose , sinuses & lungs.

Nasal mucosa contain antigen presenting cells, which are similar to dendritic langerhans cells . These cells capture and process allergens in nasal mucosa. Immunologic homing occurs when T lymphocytes are activated by antigen processing in the PNS & migrate to the mucous membranes of adjacent mucosa thereby extending the inflammatory process to the lower airway. The release of cytokines recruites other inflammatory mediators to the upper & lower airway(13,14,15).

HUMORAL PATHWAY:

The direct passage of mediators produced by activated inflammatory cells from the sinsuses through postnasal drip exerts a significant effect on bronchial responsiveness(16).

NEURAL PATHWAY:

The cholinergic pathway has a keyrole in maintaining resting bronchial muscle tone & in mediating acute bronchospastic responses(20) . Lungs are innervated by parasympathetic, non-adrenergic and noncholinergic autonomic pathway. Receptors in the nose & pharynx and presumably in the paranasal sinuses produce afferent fibers that form part of trigeminal nerve, which passes to brain stem & connects with the reticular formation of dorsal vagal nucleus. From the vagal nucleus, parasympathetic efferent fibers travel in the vagus nerve to the bronchi. Development of asthma is associated with cholinergic hyper responsiveness and a postulated partial B-adrenergic receptor blockade. Sinusitis may be associated with a reversible pre existing partial B-adrenergic blockade that improves with treatment of upper airway inflammation.

The non adrenergic , non-cholinergic nervous system of neuropeptides also contributes to lower airway autonomic function. Neuro peptides trigger smooth-muscles contraction,vasodilation plasma extravasation and mucus hypersecretion(21).

ROLE OF NITRIC OXIDE.

The nitric oxide concentration decreases during acute maxillary sinusitis. The nitric oxide level returns to base line after sinusitis is resolved. A reduction in Nitric oxide is a potent modulator of bronchial tone may precipitate bronchial hypersecretion(17).

MATERIAL AND METHODS

INCLUSION CRITERIA:

Patients referred from Asthma & Allergy clinic as a case of Bronchial asthma are screened for chronic rhinosinusitis. Cases diagnosed a chronic rhinosinusitis are treated medically for 3 weeks(23) and those refractory to conservative management are subjected to surgical management.

EXCLUSION CRITERIA:

Patients with chronic obstructive pulmonary disease, cardiac diseases, pulmonary tuberculosis, Bronchiectasis, fungal sinusitis & those who have already undergone nasal surgeries are excluded from the study.

PLACE OF STUDY : Government Stanley Medical College, Chennai.

PERIOD OF STUDY: January 2005 to June 2006.

MEASURES FOR DIAGNOSING CHRONIC RHINOSINUSITIS (31):

1.Continous symptoms that persist for 12 consecutive weeks or longer and physical findings of rhinosinusitis on examination or radiographic sinus imaging.

2.One of these signs of inflammation must be present and identified in association with on going symptoms consistent with Chronic Rhinosinusitis:

(A) Discoloured nasal discharge arising from nasal passages,nasal polyps or polypoid swelling as identified on physical examination.

(B) Edema or erythema of the middle meatus or ethmoidal bulla identified on nasal endoscopy.

(C) Generalised or localised erythema ,edema or granulation tissue . If the middle meatus or ethmoidal bulla is not involved radiologic imaging is required.

(D)Imaging modalities for confirming diagnosis

- CT SCAN demonstrating isolated or diffuse mucosal thickening,bone changes,air-fluid level.
- Plain sinus radiograph (Waters view) revealing mucous membrane thickening of 5 mm or greater or complete opacification of one or more sinuses.
- MRI is not recommended as an alternative to CT scan for routine diagnosis of Chronic rhinosinusitis because of its high sensitivity but lack of specificity.

METHOD OF SCREENING-TASK FORCE ON RHINOSINUSITISCRITERIA

Major criteria:

Facialpain/pressure**.**

Facialcongestion/fullness.

Nasalblock

Nasaldischarge

Hyposmia/anosmia

Purulence in middle meatus on anterior rhinoscopy

Fever (acute rhinosinusitis)

Minor criteria

Headache

Halitosis

Fatigue

Dentalpain

Cough

Earpain & Fever(non-acute)

For diagnosis 2major or 1 major &2 minor or purulent discharge in middle meatus alone are required(26)

X- ray paranasal sinuses waters view taken for those patients diagnosed as rhinosinusitis case and then they are subjected to diagnostic nasal endoscopy under local anesthesia. Cotton pledgets are soaked in the mixture of 10ml of 4% lignocaine with 1ampoule of 1:1000 epinephrine . The cotton pledgets are placed in the middle meatus till the end of the middle turbinate to block sphenopalantine branches, another pledget in the sphenoethmoidal area filling the space between the septum and middle turbinate . A pledget in the floor of the nasal cavity .After 10minutes, DNE was done using 0^{o} and 30 ^{o} rigid nasal endoscope .

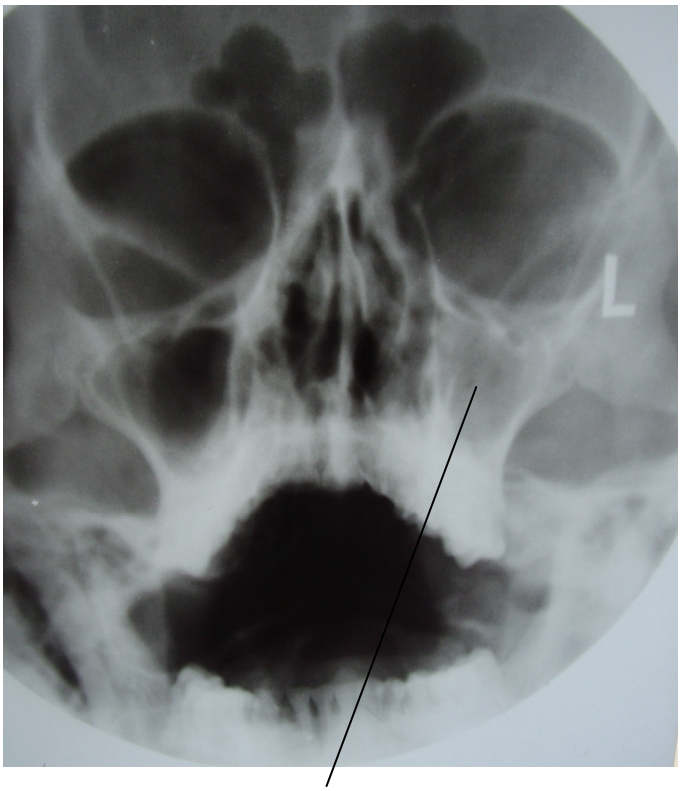
3 steps of systematic examinations in each pass was done. In First pass , endoscope is passed beneath the inferior turbinate parallel to the floor of the nose into the postnasal space. Adenoids and orifice of the eustachian tube can be seen clearly. Fossa of Rossenmuller is seen just behind & above eustachian orifice as a recess. 180^{o} rotation longitudinally in the opposite direction reveals contralateral side structures.The upper surface of the soft palate can be clearly seen as floor. Hasner's valve,guarded by one or two mucosal folds around the opening of the nasolacrimal duct is located laterally at the highest anterior point of the inferior meatus.

In Second pass,endoscope is guided past beneath the middle turbinate to the upper edge of the choana to look up into the area of sphenoethmoidal recess. It can sometimes be difficult to get a good view with 4mm scope.Superior turbinate,sometimes supreme turbinate & natural sphenoid ostium can be visualized. In Third pass endoscope is passed into middle meatus between the middle turbinate & lateral wall of the nose Uncinate process would be lateral to middle turbinate &bulla can be seen behind it.

Maxillary ostium is situated at a 15 ^{o} angle to the vertical plane anteroinferior to the lower anterior margin of bulla, in the infundibulum obscured by hooding uncinate process. Any discharge in the middle meatus, sphenoethmoidal recess, accessory ostium,polyps,polypoidal mucosa and anatomical variants like medialised uncinate, prominent agger nasi etc., are observed and recorded .

Patients are managed with antibiotics, antihistamines, decongestant nasal drops and steam inhalation for a period for 3 weeks and CT PNS taken after that. Evidence of sinusitis observed in CT Scan. Patients refractory to conservative management are subjected to surgical management .

XRAY PARA NASAL SINUSES WATERS VIEW WITH EVIDENCE OF SINUSITIS



Haziness in left maxillary sinus.

EVALUATION

Patients with Bronchial asthma & Chronic rhinosinusitis are evaluated pre-operatively and post-operatively as follows:

API GUIDELINES OF GRADING ASTHMA

Class	Diurnal symptoms	Nocturnal symptoms
Mild intermittent	<2attacks in a week asymptomatic in between.	<2attacks in a month.
Mild persistent	>2attacks in a week but <1 in a day	>2attacks in a month
moderate persistent	Daily symptoms exacerbations >2 in a week	>1 attack in a week
severe persistent	Continoussymptoms& frequent exacerbations	

Scoring system for individual symptoms of Asthma and Rhinosinusitis:

0-No symptoms

1-mild symptoms, normal activities not affected.

2-moderate symptoms, slight disturbance in normal activities

3-severe symptoms, serve disturbance in normal activites

4-very severe symptoms unable to work.

Asthma symptoms:Dyspnea & Wheeze.

Sinusitis symptoms:Nasal discharge, Hyposmia, Facialpain,Headache& Nasal block

PEAK EXPIRATORY FLOW RATE (PEFR)

Expiratory peak flow (PEF) is the maximum flow generated during expiration performed with maximal force and started after a full inspiration . PEF is appreciably larger if the maneuver is performed without pause immediately after the inspiration than if it is performed after a pause . Contrary to popular belief PEF is not effort dependent if the maneuver is performed sufficiently forcefully in most subjects PEF is determined by flow limitation in central, possibly also in more peripheral airways .

Report the largest value of 3 correctly performed maneuvers, but the difference between the largest two should be less than 40 L/min; if the difference is larger then have up to 2 extra efforts performed. If even then the two largest values differ by > 40 L/min, then report the largest one with a note to the effect that reproducible measurements could not be obtained. The best reference value for a subject is produced when he is in a clinically good condition.

PEF is not often used to assess bronchodilator responsiveness. An increase in PEF by 60 L/min or more in adults has been regarded as a sign of significant improvement An abnormally low PEF may be due to obstructive lung disease,unsatisfactory cooperation, prior inhalation not to total lung capacity restricted lung expansion (stiff chest cage, muscular or neurogenic disorder).

EXPECTED FORMULA FOR PEFR.

FOR MALES.

Age in yrs	7 – 15 yrs	16 – 40 yrs	40 yrs <
Formula	(5 x ht.in cms) – 420	(2.74 x ht.in cms) + 53.4	567 – (2.24 x age)
Normal range	80-450 L/min	400 –650 L / min	300 – 500L/min.

FOR FEMALES.

Age in yrs	7 – 15 yrs	16 – 40 yrs	40 yrs <
Formula	(5 x ht.in cms) – 430	(4.65 x ht.in cms) – 360	438 – (2.24 x age)
Normal range	70-400 L/min	250 –400 L / min	200 – 400L/min.

Grading asthma on the basis of PEFR

PEAK EXPIRATORY FLOW RATE	GRADE OF ASTHMA
>80% of expected PEFR	MILD
60-80% of expected PEFR	MODERATE
<60% of expected PEFR	SEVERE

PORTABLE WRIGHTS PEAK FLOW METER.

A PATIENT USING PEAK FLOW METER.

107 patients (88 females & 19 males) with age distribution of (10– 70 yrs) are screened for chronic rhino sinusitis using TASK FORCE RHINOSINUSITIS CRITERIA(Lanza&Kennedy). 53 pts clinically diagnosed to have rhinosinusitis are further evaluated using Diagnostic nasal endoscopy and x-ray of paranasal sinuses waters view. 47 cases diagnosed as having chronic rhinosinusitis are treated with antibiotics, antihistamines, decongestant nasal drops, steam inhalation for a period of 3wks(23) and CT scan taken after medical treatment . 26 patients found to have abnormality in CT PNS after medical treatment are considered to be refractory to conservative management and subjected to functional endoscopic sinus surgery.

Likert symptom scale for asthma & sinus symptoms are taken before and after surgery. PEFR is used to assess the lung function of patients periodically (1stmonth,2ndmonth&3rdmonth) after treatment .Functional endoscopic sinus surgery was performed under general anaesthesia. Pre operatively injection Deriphylline and injection Dexamethasone were given intravenously. Considering the possiblity of bronchospasm due to anxiety,aspiration of secretions and blood in local anaesthesia, general anaesthesia is preferred.

Group 1 : Those Patients with asthma and rhinosinusitis managed surgically(n – 26).

Group 2: Patients with asthma and rhinosinusitis managed conservatively (n - 21).

Group 3 : Patients with asthma alone selected randomly as controls(n- 30).

STATISTICAL ANALYSIS OF RESULTS.

ANALYSIS OF PATIENTS IN GROUP 1

Patients Sl no.	Age in yrs / sex	Height in cms.	Normal PEFR L/min.	Pre-operative PEFR L/min.	Post-operative PEFR L/min.	%of improvement in PEFR
1	40/F	140	291	200 (68.7%)	300(103.09%)	34.39
2	19/F	142	300	200 (66.6%)	300 (100.%)	33.4
3	35/F	145	314	250 (79.6%)	400(127.38%)	47.78
4	23/F	140	291	250 (85.9%)	350 (120%)	34.1
5	20/F	140	291	200 (68.7%)	300 (103%)	34.3
6	44/F	150	340	200 (58.8%)	300 (88.23%)	29.43
7	33/F	150	338	250 (73.96%)	400(118.34%)	44.38
8	30/F	140	291	250 (85.91%)	350 (120.2%)	34.29
9	38/F	158	365	200 (54.79%)	400(109.59%)	54.8
10	16/F	150	338	200 (59.17%)	400(118.34%)	59.17
11	40/F	142	300	200 (66.66%)	350(116.66%)	50
12	24/F	156	365	250 (68.49%)	450(123.28%)	54.79
13	33/F	145	314	250 (79.6%)	400(127.38%)	47.78
14	30/F	140	291	250 (85.9%)	350 (120%)	34.1
15	38/F	142	300	200 (66.66%)	350(116.66%)	50
16	36/F	150	338	200 (59.17%)	400(118.34%)	59.17
17	40/F	142	300	200 (66.66%)	400(133.33%)	66.73
18	30/M	150	464	300 (64.65%)	500 (107.7%)	43.05
19	43/F	140	291	200 (68.7%)	350 (120.2%)	51.5
20	27/F	142	300	250 (83.33%)	400 (133.3%)	49.97
21	27/F	150	338	200 (59.17%)	450(133.13%)	73.96
22	40/F	164	402	200 (49.75%)	400 (99.5%)	49.75
23	26/F	140	291	200 (68.7%)	300(103.09%)	34.39
24	39/F	145	314	200 (63.69%)	300 (99.5%)	35.85
25	25/F	142	300	200 (66.6%)	400(133.33%)	66.73
26	40/F	142	300	200 (66.6%)	300(103.09%)	36.49

Total average of improvement in PEFR after surgical management 46. 55 %

ANALYSIS OF PATIENTS IN GROUP 2

Patients Sl no.	Age in yrs / sex	Height in cms.	Normal PEFR L/min.	Pre-operative PEFR L/min.	Post-operative PEFR L/min.	%of improvement in PEFR
1	20/F	142	300	200 (66.6%)	300 (100%)	33.4
2	37/M	160	492	250 (50.8%)	350 (71.14%)	20.34
3	15/M	156	360	200 (55.5%)	300 (83.3%)	27.8
4	55/F	150	317	200 (63.1%)	250 (78.86%)	15.7
5	30/M	146	453	300 (66.225%)	400 (88.3%)	22.07
6	50/M	150	455	250 (54.94%)	350 (76.9%)	21.96
7	25/F	142	300	200 (66.6%)	250 (83.33%)	16.73
8	27/M	146	453	300 (66.23%)	450 (99.3%)	33.07
9	38/F	140	291	150 (51.5%)	250 (85.9%)	34.4
10	45/F	150	317	200 (63.1%)	250 (78.9%)	15.8
11	17/F	142	300	150 (50%)	250 (83.3%)	33.3
12	54/F	150	317	200 (63.1%)	300 (94.63%)	31.53
13	34/F	142	300	250 (83.3%)	300 (100%)	16.7
14	30/F	150	338	200 (59.17%)	300 (88.75%)	29.58
15	25/F	140	291	200 (68.7%)	250 (85.91%)	17.2
16	18/F	142	300	200 (66.6%)	300 (100%)	33.4
17	30/F	146	319	250 (78.8%)	350 (109%)	30.2
18	27/F	140	291	200 (68.7%)	250 (85.9%)	17.2
19	23/F	142	300	250 (83.3%)	300 (100%)	16.7
20	37/F	140	291	200 (68.7%)	250 (85.9%)	17.2
21	51/M	150	455	250 (54.9%)	400 (87.9%)	33

Total average of improvement in PEFR after surgical management 24.63 %

ANALYSIS OF PATIENTS IN GROUP 3

Patients Sl no.	Age in yrs / sex	Height in cms.	Normal PEFR L/min.	Pre-operative PEFR L/min.	Post-operative PEFR L/min.	%of improvement in PEFR
1	23/F	140	291	200 (68.7%)	200 (68.7%)	0
2	52/F	152	321.52	150 (46.65%)	200 (62.22%)	15.57
3	38/F	142	300	200 (66.6%)	250 (83.33%)	16.67
4	20/F	146	319	200 (62.69%)	200 (62.69%)	0
5	25/M	146	453.44	300 (66.16%)	300 (66.16%)	0
6	38/F	150	337.5	200 (59.25%)	200 (59.25%)	0
7	22/M	150	464.4	300 (64.59%)	250 (53.83%)	-10.76
8	20/F	140	291	150 (51.54%)	150 (51.54%)	0
9	40/F	142	300	250 (50%)	200 (66.66%)	16.66
10	42/F	140	343.9	200 (58.15%)	200 (58.15%)	0
11	40/F	148	328.2	200 (60.94%)	250 (76.17%)	15.23
12	20/M	145	450.7	300 (66.56%)	300 (66.56%)	0
13	45/F	143	337.2	150 (44.5%)	200 (59.31%)	14.81
14	20/F	140	291	150 (51.55%)	250 (85.91%)	34.36
15	40/F	164	402.6	200 (49.67%)	300 (74.51%)	24.87
16	29/F	158	374.7	150 (40%)	200 (53.37%)	13.37
17	30/F	150	337.5	200 (59.25%)	200 (59.25%)	0
18	29/F	140	291	150 (51.54%)	150 (51.54%)	0
19	35/F	146	453.4	300 (66.16%)	300 (66.16%)	0
20	36/F	142	300	200 (66.66%)	250 (83.33%)	16.67
21	30/F	141	295.65	200 (67.64%)	200 (67.64%)	0
22	32/F	145	314.25	150 (47.73%)	200 (63.64%)	15.91
23	44/F	147	339.44	200 (58.02%)	250 (73.65%)	14.73
24	38/F	141	295.65	250 (84.55%)	200 (67.64%)	-16.91
25	30/F	158	374.7	250 (66.72%)	300 (80.06%)	13.34
26	42V	143	343.92	300 (87.22%)	300 (87.22)	0
27	35/F	150	337.5	200 (59.25%)	200 (59.25%)	0
28	14/F	138	260	150 (57.69%)	200 (76.92%)	19.23
29	59/F	141	305.84	200 (65.39%)	200 (65.39%)	0
30	46/F	147	334.96	200 (59.70%)	250 (74.6%)	14.93

Total average of improvement in PEFR in control group = 7.29 %

STANDARD DEVIATION OF MEAN VALUE OF GROUP 1

Sl.no	%of improvement in PEFR x_1	Arithmetic mean x_2	Deviation from mean($x_{1-} x_{2}$)	$(x_1-x_{2})^2$
1	34.39	46.55	-12.16	147.87
2	33.4	46.55	-13.15	172.92
3	47.78	46.55	1.23	1.51
4	34.1	46.55	-12.45	155
5	34.3	46.55	-12.25	150.06
6	29.43	46.55	-17.12	293.09
7	44.38	46.55	-2.17	4.70
8	34.29	46.55	-12.26	150.30
9	54.8	46.55	8.25	68.06
10	59.17	46.55	12.62	159.26
11	50	46.55	3.45	11. 90
12	54.79	46.55	8.24	67.89
13	47.78	46.55	1.23	1.51
14	34.1	46.55	-12.45	155
15	50	46.55	3.45	11.90
16	59.17	46.55	12.62	159.26
17	66.73	46.55	20.18	407.23
18	43.05	46.55	-3.50	12.25
19	51.5	46.55	4.95	24.50
20	49.97	46.55	3.42	11.69
21	73.96	46.55	27.41	751.30
22	49.75	46.55	3.20	10.24
23	34.39	46.55	-12.16	147.86
24	35.85	46.55	-10.7	114.49
25	66.73	46.55	20.18	407.23
26	36.49	46.55	10.06	101.20

$$\sqrt{\frac{(x_1-x_{2})^2}{n_1-1}} = \sqrt{\frac{3698.16}{25}}$$

STANDARD DEVIATION OF MEAN VALUE GROUP I = 12.16

STANDARD DEVIATION OF MEAN VALUE OF GROUP 2

Sl.no	% of improvement in PEFR y_1	Arithmetic mean y_2	Deviation from mean$(y_1 - y_2)$	$(y_1-y_2)^2$
1	33.4	24.63	8.77	76.91
2	20.34	24.63	-4.29	18.40
3	27.8	24.63	3.17	10.04
4	15.7	24.63	-8.93	79.74
5	22.07	24.63	-2.56	6.55
6	21.96	24.63	-2.67	7.13
7	16.73	24.63	-7.9	62.41
8	33.07	24.63	8.44	71.23
9	34.4	24.63	9.77	95.45
10	15.8	24.63	-8.83	77.97
11	33.3	24.63	8.67	75.17
12	31.53	24.63	6.9	47.61
13	16.7	24.63	-7.93	62.88
14	28.58	24.63	3.95	15.60
15	17.2	24.63	-7.43	55.2
16	33.4	24.63	8.77	76.91
17	30.2	24.63	5.57	31.03
18	17.2	24.63	-7.43	55.2
19	16.7	24.63	-7.93	62.88
20	17.2	24.63	-7.43	55.2
21	33	24.63	8.37	70.06

$$\sqrt{\frac{(y_1-y_2)^2}{n_2-1}} = \sqrt{\frac{1113.57}{20}}$$

STANDARD DEVIATION OF MEANVALUE IN GROUP 2 = 7.46

STANDARD DEVIATION OF MEAN VALUE OF GROUP 3.

Sl.no	% of improvement in PEFR z_1	Arithmetic mean z_2	Deviation from mean(z_1-z_2)	$(z_1-z_2)^2$
1.	0	7.29	-7.29	53.14
2.	15.57	7.29	8.28	68.56
3.	16.67	7.29	9.38	87.98
4.	0	7.29	-7.29	53.14
5.	0	7.29	-7.29	53.14
6.	0	7.29	-7.29	53.14
7.	-10.76	7.29	18.05	325.8
8.	0	7.29	-7.29	53.14
9.	16.66	7.29	9.37	87.79
10	0	7.29	-7.29	53.14
11	15.23	7.29	7.94	63.04
12	0	7.29	-7.29	53.14
13	14.81	7.29	7.52	56.55
14	34.36	7.29	27.07	732.78
15	24.87	7.29	17.58	309.05
16	13.37	7.29	6.08	36.96
17	0	7.29	-7.29	53.14
18	0	7.29	-7.29	53.14
19	0	7.29	-7.29	53.14
20	16.67	7.29	9.38	87.98
21	0	7.29	-7.29	53.14
22	15 .91	7.29	8.62	74.3
23	14.73	7.29	7.44	55.35
24	-16.91	7.29	24.2	585.64
25	13.34	7.29	6.05	36.6
26	0	7.29	-7.29	53.14
27	0	7.29	-7.29	53.14
28	19.23	7.29	11.94	142.56
29	0	7.29	-7.29	59.14
30	14.93	7.29	7.64	58.36

$$\sqrt{\frac{(z_1-z_2)^2)}{n-1}} = \sqrt{\frac{3553.26}{30-1}}$$

$$= \sqrt{122.53}$$

STANDARD DEVITION OF MEAN VALUE OF GROUP 3 = 11.07.

Pooled standard deviation between group 1 & group 3 (SD_P) = $\sqrt{\frac{(X_1-X_2)^2}{(N_1-1)} + \frac{(Z_1-Z_2)^2}{(n_3-1)}}$

=16.44

Applying Students t' Test;

t ' value for group 1&3 = $\frac{\mathbf{X - Z}}{SD_P\sqrt{\frac{1}{N_1} + \frac{1}{N_3}}}$ = $\frac{46.55-7.29}{16.44 \times .26}$ =9.2

Degree of freedom =(26+30-2) =54

P value for the degree of freedom 54 is < .001 and it is significant.

Pooled standard deviation between group 2&3 (SD_P) = $\sqrt{\frac{(Y_1-Y2)^2}{N_2-1} + \frac{(Z_1-Z_2)^2}{N_3-1}}$

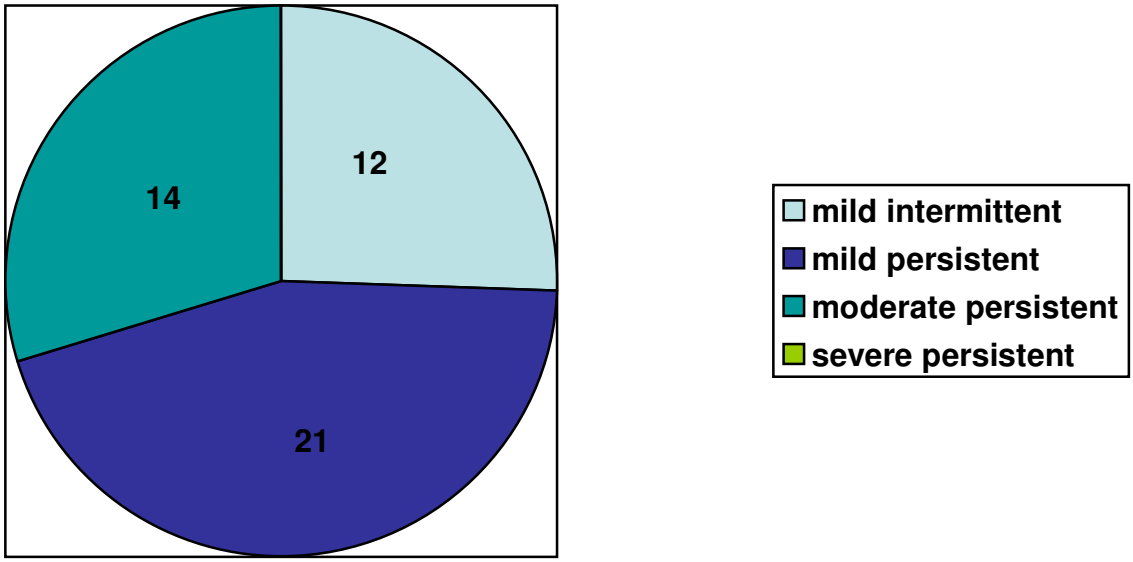
=13.4

t ' value for group 2 &3 = $\frac{\mathbf{Y-Z}}{SD_P\sqrt{\frac{1}{N_2} + \frac{1}{N_3}}}$ = $\frac{24.63-7.29}{13.4 \times .29}$ = 4.62

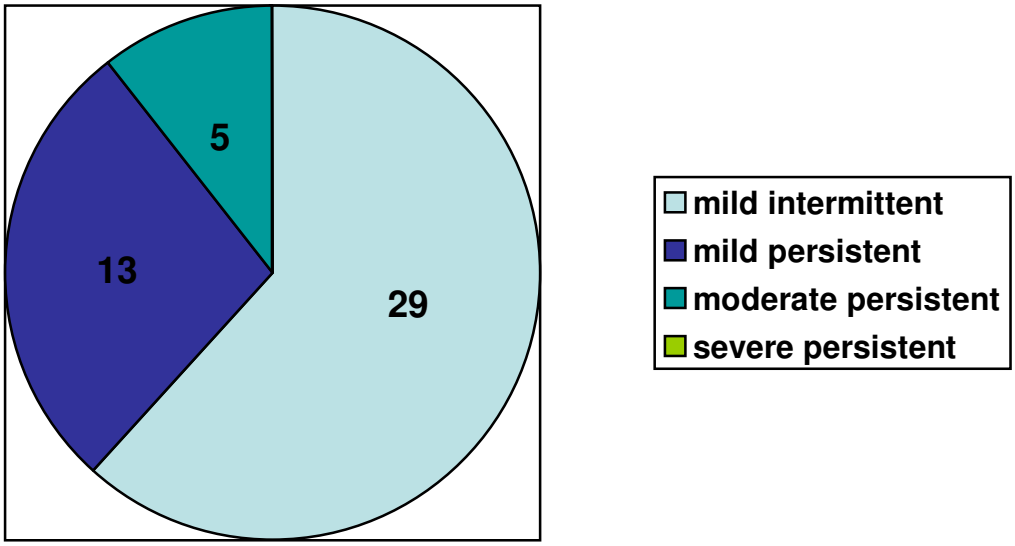
Degree of freedom = (21+30-2)= 49.

P value for the degree of freedom 49 is < .001 & it is significant.

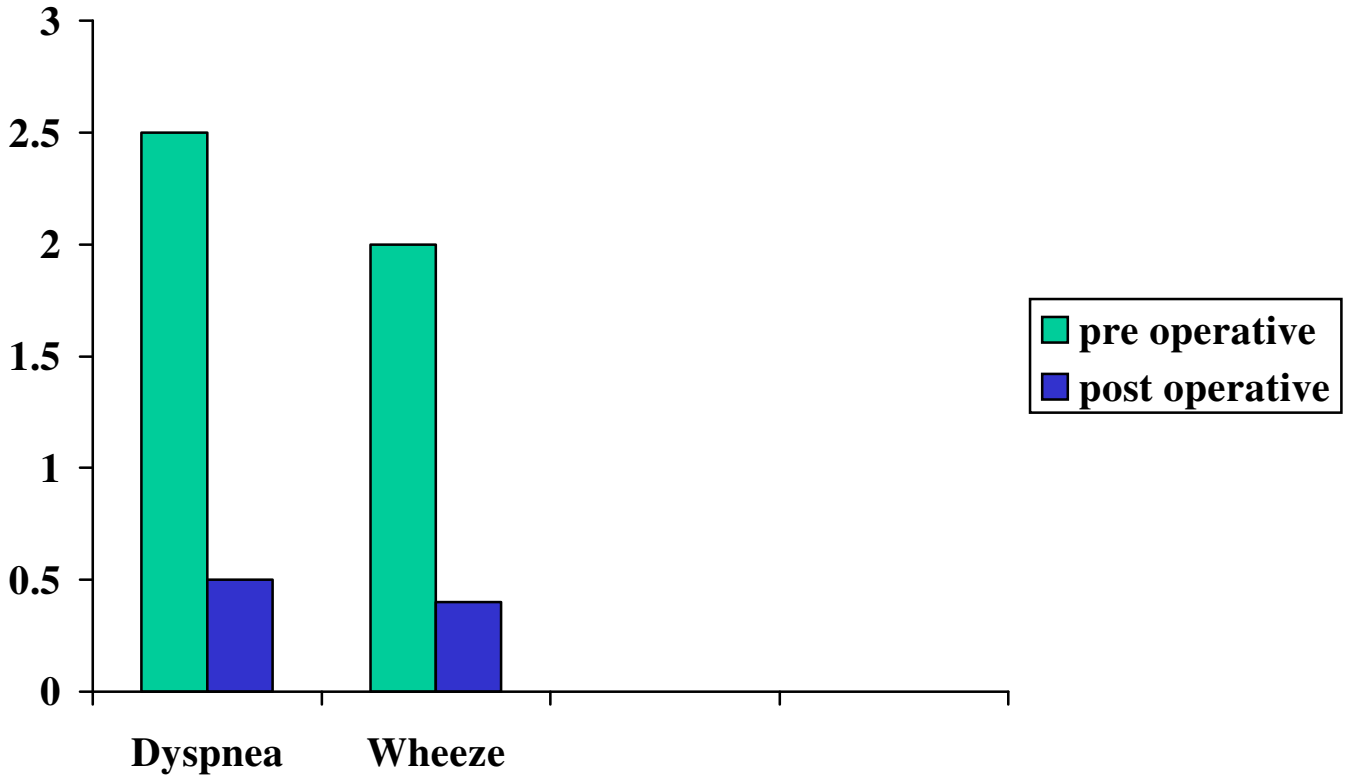
ASTHMA GRADING BEFORE TREATMENT



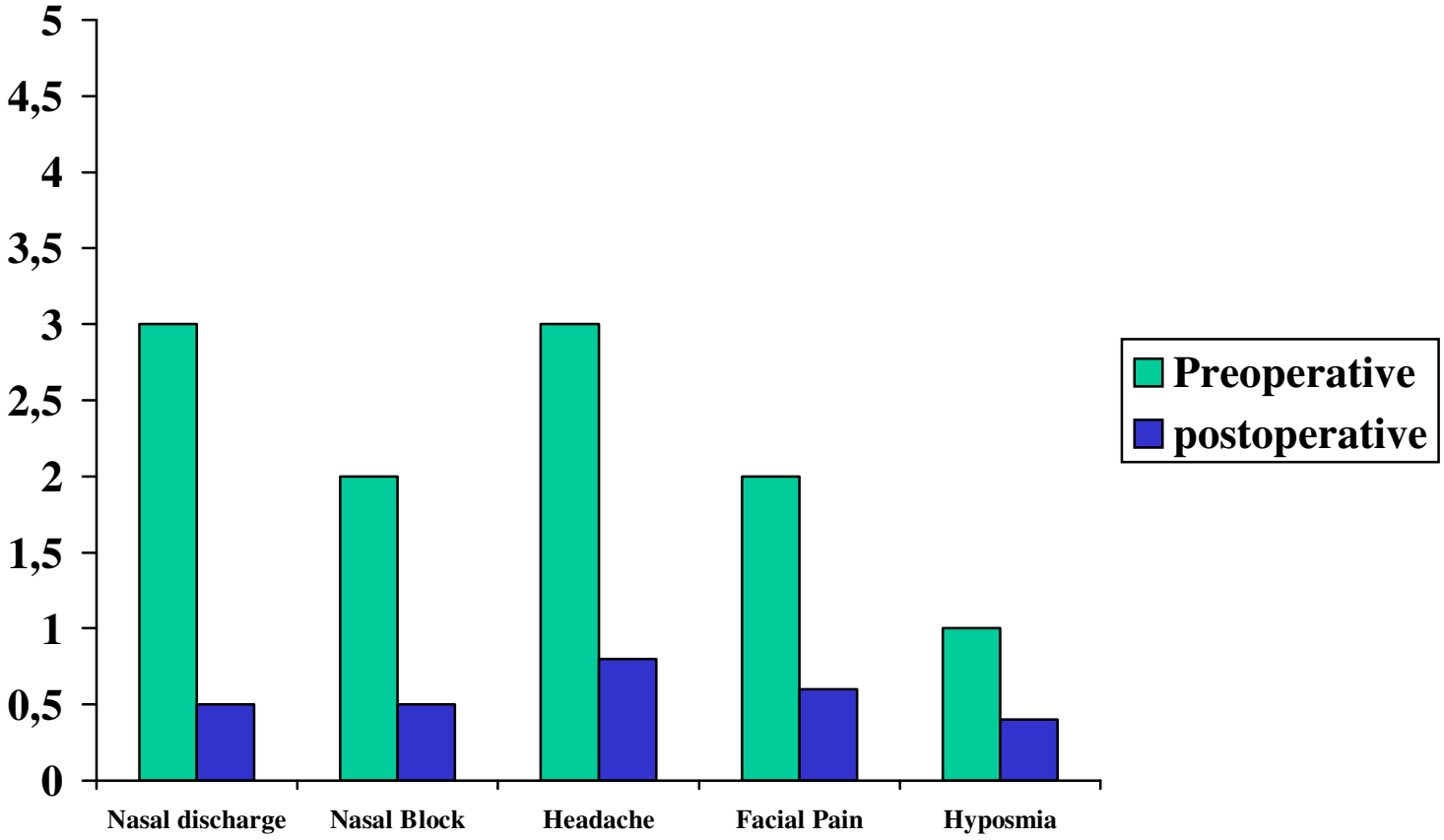
ASTHMA GRADING AFTER TREATMENT



Changes in symptoms of Asthma



Changes in symptoms of rhinosinusitis



DISCUSSION

In the past,surgeons hesitated to perform surgical treatment for chronic rhinosinusitis in asthmatic patients because surgery for chronic rhinosinusitis with coexisting asthma was thought to hinder the improvement of asthma symptoms further aggravate bronchial asthma. Since the 1980's , however,several authors have reported that medical or surgical treatment of rhinosinusitis, although no causal relationship can be clearly determined,several findings and evidence show improvement in the clinical course of bronchial asthma in adult and pediatric asthmatic patients with chronic rhinosinusitis after medical or surgical treatment .

Since the publication of Messerklinger's classic work,which demonstrated an improved understanding of the pathogenesis of rhinosinusitis, ESS has emerged as the surgical procedure of choice for treating chronic rhinosinusitis that is refractory to medical management . A survey of the literature reveals a number of studies reporting on the effects of ESS on the course of asthma in asthmatic patients with chronic rhinosinusitis. In this study asthma severity, frequency of attacks, and pulmonary function were all improved.

In our study among 107 patients, 47 had sinusitis.After surgical management the average improvement in PEFR is 46.55 & after conservative management average improvement in PEFR is 24. 63 which when compared to the control group is significant. The follow up period is for three months . In our study we utilised both objective and subjective parameters to assess the effect of management of rhinosinusitis in the clinical course of bronchial asthma.

This study demonstrates that a combination of FESS, careful postoperative care, and appropriate medical therapy for chronic rhinosinusitis has a favorable effect on asthma in patients with symptomatic chronic sinusitis.

LITERATURE REVIEW

In the Department of Otorhinolaryngology Head and Neck Surgery, University of Pennsylvania Medical Center, Philadelphia, USA using objective and subjective criteria, a study was performed by Senior & Kennedy(18) to assess the long-term impact of functional endoscopic sinus surgery (FESS) in patients with chronic rhinosinusitis and asthma at an average follow-up of 6.5 years. One hundred and twenty patients who underwent FESS for chronic rhinosinusitis were followed. Seventy-two (60%) patients responded to a follow-up questionnaire, and 30 (42%) of them reported a history of asthma. Subjective levels of improvement and assessments of medication need were evaluated and statistically assessed with parametric and nonparametric methods. Of these 30 patients, 27 (90%) reported that their asthma was better than it had been before FESS. Average reported improvement increased from 49% at 1.1 years after surgery to 65% at 6.5 years after surgery. Asthma attacks declined in 20 of 27 (74.1%). Medication use for asthma showed similar improvement, with approximately half reporting less inhaler usage and nearly two thirds reporting less oral steroid use.

In the Department of Surgery, University of Missouri-Columbia, by Cook & Nishioka(4). Twenty asthma patients who underwent functional endoscopic sinus surgery for chronic sinusitis were studied. Medical records and questionnaire data for these 20 patients were studied regarding the impact of sinus disease and functional endoscopic sinus surgery on their asthma .95% reported that their asthma was worsened by their sinus disease and 85% reported that functional endoscopic sinus surgery improved their asthma .Of the 13 patients who used both inhalers and systemic medication, 53.8% were able to eliminate some of their medication .Furthermore, 61.5% of these patients had a concomitant reduction in their inhaler use .All patients (six) who used only inhalers experienced a reduction in their inhaler use, and two patients were able to eliminate their inhalers completely. One of two patients who were steroid dependent was able to discontinue steroids after surgery. Of patients who used steroids intermittently (13), 53.8% were able to eliminate the use of steroids after surgery Patients who required preoperative hospital admissions (4) and emergency room or urgent physician office visits (18) had a 75.0% and 81.3% ($p < 0.001$) reduction in visits, respectively, after surgery. Because 43% of the cost of asthma is the result of hospitalizations and emergency department/urgent physician office visits, a significant impact on health care costs can be realized with functional endoscopic sinus surgery in this patient population.

A study was carried out at the Institute of Allergy and the Department of otolaryngology of Shanxi Medical University. Patients with both CRS and asthma were considered to be enrolled into this study. Thirty-eight patients with both CRS and chronic asthma were enrolled to this study. A group of 25 patients with CRS alone were enrolled into this study served as controls. All patients required surgical treatment since they had tried different medical remedies and had not obtained satisfactory results. These 38 patients also suffered from perennial asthma. All of them clearly stated treatment since they had tried different medical remedies and had not obtained satisfactory results. Their symptoms could be controlled by inhaling β2-adrenoreceptor agonists, or theophyline preparations. Lung function tests were performed for patients with asthma before and 2 months after sinus surgery. A group of 23 patients with asthma alone were enrolled into this study . Clinical symptoms of patients with CRS-asthma and CRS alone were improved after FESS.Two months after the FESS, FEV1 value fall-percentage in response to antigen challenge was also improved in CRS-asthma patients improvement >12% could be considered as positive results), but not in patients with asthma alone .The post-FESS clinical asthma scores decreased significantly the clinical symptoms of CRS, such as nasal blockage and nasal purulent secretions were also assessed by patients. The FESS resulted in markedly attenuating the CRS clinical scores clinical study of endoscopic sinus surgery for sinusitis in patients with bronchial asthma served as asthma controls. Clinical symptoms of patients with CRS-asthma and CRS alone were improved after FESS.

Rachelefsky(1) et al were early observers of worsening of lower airway disease with sinusitis. 48 children with asthma were identified to have chronic daytime and night time symptoms of cough and wheeze. Water's view of paranasal sinuses revealed abnormalities in all the 48 children (mucosal thickening, maxillary sinus opacification (or) air fluid levels) all the children treated with antibiotics. At the completion of treatment , nearly 80% had normal radiographs of paranasal sinuses. Of the tested, 67% had normal pulmonary functions. This study provides some evidence that sinusitis can affect lower airway disease.

Studies of adult patients by Slavin & Slavin(2) focused on corticosteroid dependent asthmatics with chronic rhinosinusitis not responding to medical management . Subsequent surgical intervention created significant improvement in chronic asthma in two thirds of the patient.

Ramsdale(9) et al (1985) used methacholine and isocapnic hyperventilation to measure bronchial hyper responsiveness in 25 subjects with rhinitis who didn't have the diagnosis of bronchial asthma. In six subjects , the provocative concentration of the methacholine that decreased the FEV_1 by 20% was less than 8mg/ml in a range of responsiveness found in asthma. There were four additional subjects whose Provocative concentration was in 8-16mg/ml range . The observation of ramsdale etal also indicate hyper responsiveness of lower airway in subjects with rhinitis.

Bellofiore(9) et.al (1987) used sensitized rats to assess the contribution of upper and lower airways to the changes in pulmonary resistance after inhalation of antigen. After inhalation of antigen through nose to the upper and lower airway resistance increased. When the antigen was given via nasal route both upper and lower airway resistance increases. In contrast when the antigen was introduced through tracheostomy lower airway resistance increased but not the upper airway. To explore the possible mechanism by which upper airway allergic response enhanced lower airway function,the animals pretreated with inhaled atropine. Atropine blocked the increase in lower airway resistance following application of antigen these observation suggest that reaction of upper & lower respiratory tract are mediated by cholinergic mechanisms.

Brugman(22) and co-workers developed a rabbit model to identify mechanisms associated with lower airway changes in sinusitis. Rabbits have well developed maxillary sinuses which are accessible and lower airway physiology measured . Maxillary sinuses were then injected with the active complement component C5a to cause a inflammatory process. Following C5a injection rabbits developed bronchial responsiveness. Animals received C5a in knee bursa to determine whether an lower airway changes. Rabbits were intubated prior to C5a injection, injected but kept in the head down position (or) in upright position.Animals in upright position and not intubated noted to have bronchial hyperresponsiveness. These observation suggests that drainage of material to the lower airway may contribute to changes in airway function.

Hun-Jong Dhong,Seoul, performed a study on the effect of endoscopic sinus surgery on asthmatic patients with chronic rhinosinusitis. They performed a study to evaluate the effectiveness of FESS in patients with asthma who underwent FESS for rhinosinusitis. The use of antiasthma medication and postoperative asthma symptoms was analyzed. Objective changes of pulmonary function tests were evaluated. There was a significant improvement in diurnal and nocturnal asthma symptoms. Improvements in asthma medication scores were also confirmed, and individual asthma symptoms(dyspnea, cough,wheezing, and sputum production) improved significantly. Despite a reduction in use of antiasthma medication after FESS,the parameters of the pulmonary function tests did not change. Both subjectively and objectively, it seems that FESS, when used to treat asthmatic patients with chronic rhinosinusitis, can play a significant role in the clinical improvement of asthma.

CONCLUSION

- All the bronchial asthma patients with nasal symptoms should be evaluated by Otorhinolaryngologist for Rhinosinusitis screening & may be included in their management protocol.

- Treating the rhinosinusitis improves the peak expiratory flow rate in asthma patients.

- Managing rhino sinusitis alleviates the asthma symptoms and improve their quality of life .

BIBLIOGRAPHY

1. Rachelefsky GS, Katz rm, Siegel SC. Chronic sinus disease with associated reactive airway disease in children .Pediatrics 1984;73:526-30.
2. Slavin RG. Relationship of nasal disease and sinusitis to asthma. Annal of Allergy 1982;49:76-80.
3. Kaufman J, Wright GW. The effect of nasal and nasopharyngeal irrigation on airway resistance in man. American Review of Respiratory Diseases 1969;100:626-30.
4. Nishioka GJ,Cook PR,Davis WE, et al. Functional endoscopic sinus surgery in patients with chronic sinusitis and asthma.Otolaryngology Head Neck Surg 1994;110:494-500.
5. Senior BA,Kennedy DW, Tanabodee J, et al . Long-term impact of functional Endoscopic sinus surgery on asthma. Otolaryngology Head Neck Surg 1999;121:66.
6. Senior BA, Kennedy DW,Management of sinusitis in the asthmatic patient Annal of Allergy Asthma Immunology1996;77:6-19.
7. Goldstein MF,Grundfast SK Dunsky EH,et al. Effect of functional endoscopic sinus surgery on bronchial asthma outcomes. Archives Otolaryngol ogy Head Neck Surgery 1999;247:63-76.

8. Global intiative for asthma. Global strategy for asthma management and prevention, NHLBI/WHO workshop report 1996.
9. Ramsdale EH, Morris MM, Robert, Asymptomatic Bronchial Hyperresonsiveness in Rhinitis. Journal of Allergy & Clinical immunology 1985;75:573-577.
10. Bellofiore S, DiMara GU,Martin JG, Changes in upper and lower airway resistance after inhalation of antigen in sensitized rats. American journal of respiratory diseases 1987;136:363-368.
11. Guyton, Textbook of physiology , pulmonary ventilation chapter 37:475-477
12. de Benedictis FM Rhinosinusitis and asthma:epiphenomenon (or) causal association ? chest 1999; 115(2):550-6.
13. Kaliner MA, OsguthorpeJD, FiremanP et al Sinusitis: bench to bedside, current findings, future directions.Otolaryngology Head & Neck Surgery 1997:116:S1-20
14. Orecchia AC, Azar MM. Exploring the inflammatory process in bronchial asthma; new developments and challenges. Bio Medicina 1998; (7):255-8.
15. Mygind N, Dahl R, Nielsen LP. Effect of nasal inflammation and intranasal anti inflammatory treatment on bronchial asthma. Respiratory Medicine 1998;92(3) 547-9
16. Irvin C.G. Sinusitis and asthma; an animal model. Journal Allergy &Clinical immonology 1992;90 521-33.
17. Baraldi E, Azzolin NM, BibanP, et al. Effect of antibiotic therapy on nasal nitric oxide concentration in children with acute sinusitis, American journal of respiratory medicine 1997;155(5) 1680-3.

18. Senior BA Kennedy DW, TanabodeeJ .et al Longterm impact of funtional endoscopic sinus surgery on Asthma Otolaryngology Head & Neck Surgery 1999;121(1): 66-8.
19. Ikeda K, Tanno N, Tamura G, et al. Endoscopic sinus surgery improves pulmonary function in patients with asthma associated with chronic sinusitis. Annal of Otorhino laryngology 1999 ;108(4): 355-9.
20. Slavin RG. Complications of allergic rhinitis: implications for sinusitis and asthma Journal of Allergy and Clinical immunology 1998;101:S357-60.
21. Rowe jones JM.The linkbetween the nose and lung ,perennial rhinitis and asthma-is it same disease? Allergy 1997;52(36 supplement):20-8.
22. Brugman SM,Larsen GC,Henson PM,Irvin CG.Increased lower airways responsiveness associated with sinusitis in a rabbit model. American review of respiratory diseases 1993;147:314-320.
23. Itzhak Brook , Microbiology and antimicrobial management of sinusities. Otolaryngology Clinics of North Amercia 2004;34:264.
24. Park. Textbook of preventive & social medicine 17th edition ;ch18:615.
25. Hun-Jong Dhong, Yoo Seok Jung, Effect of Endoscopic sinus surgery on asthmatic patients with chronic rhinosinusitis. Otoloryngology Head & Neck Surgery 2001;154:99-104.
26. Lanza DC, Kennedy DW, Adult Rhinosinusitis Defined Otolaryngology Head & neck Surgery 1997;117:S1 S7.
27. William F Jackson,John Rees ,Asthma current perspectives.1996;16-23.

28. Heinz Stammberger, Functional Endoscopic Sinus Surgery Messerklinger Technique ;1991, chapter2,15-45.

29. Jack B. Anon,James Zinreich ,Anatomy of Paranasal Sinuses .

30. Scott Brown Basic Sciences Vol 1997 1/5/14-20.

31.Classification and Management of Rhinosinusitis and its Complications.Lana.L.Jackson Otolaryngology Clinics Of North America 38 (2005) 1143-1153.

Screening proforma

Name age/sex occupation

Address

Nasal block

Post nasal discharge

Hyposmia/anosmia

Facialpain / pressure

Facial fullness / congestion

Sneezing

Bleeding per nose

Change in voice

Headache

Halitosis

Dental pain

Cough

Fatigue

Ear pain/pressure

Fever

Duration of bronchial asthma

Frequency of attacks, diurnal nocturnal

Preeipitating factors

Medications

Family h/o of asthma/allergy

H/o Tuberculosis/cardiac disease/DM/HT/STD/ Previous nasal surgeries
H/o Smoking / alcohol intake /snuff usage/betel nut chewing

Examination of nose
External contour columella
Septum

	Right	Left
Floor of nasal cavity		
Inferior meatus		
Inferior turbinate		
Middle meatus		
Middle turbinate		
Post nasal		
Coldspatula test		

Ear
Throat/neck
Respiratory system CVS

DIAGNOSTIC NASAL ENDOSCOPY FORM

GOVERNMENT STANLEY MEDICAL COLLEGE, CHENNAI.

	Right	Left
Inferior meatus		
Inferior turbinate		
Agger nasi		
Uncinate		
Middle meatus		
Middle turbinate		
Spheno ethmoidal recess		
Turbinoseptal classification		
Mucosa		
Nasopharynx		

MUCOCILIARY CLEARANCE IN SINUSES

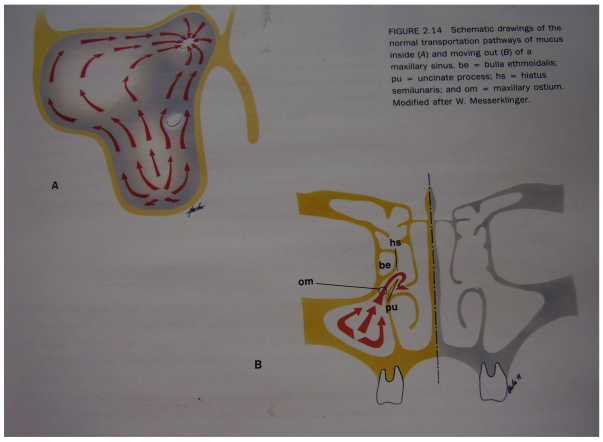
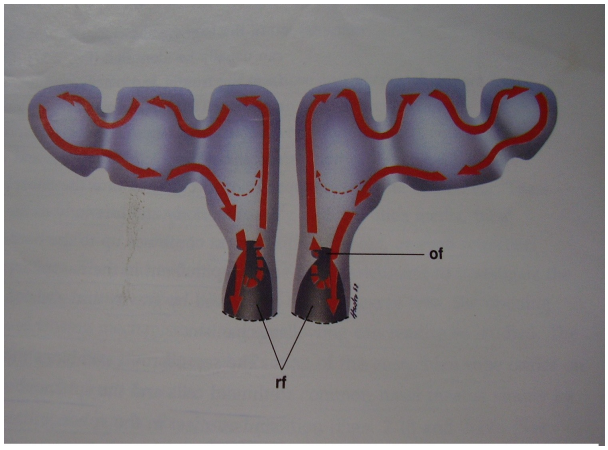


FIGURE 2.14 Schematic drawings of the normal transportation pathways of mucus inside (A) and moving out (B) of a maxillary sinus. be = bulla ethmoidalis; pu = uncinate process; hs = hiatus semilunaris; and om = maxillary ostium. Modified after W. Messerklinger.



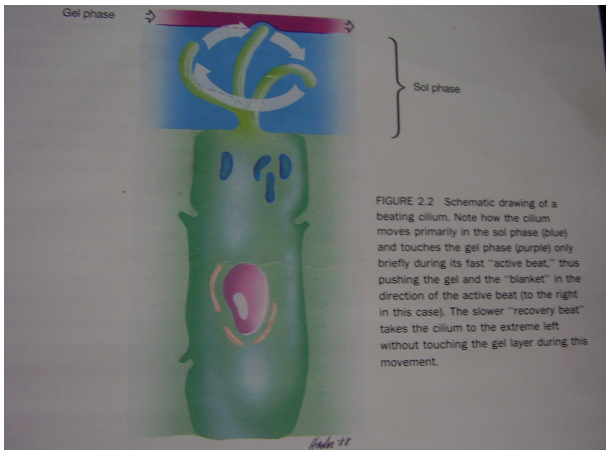
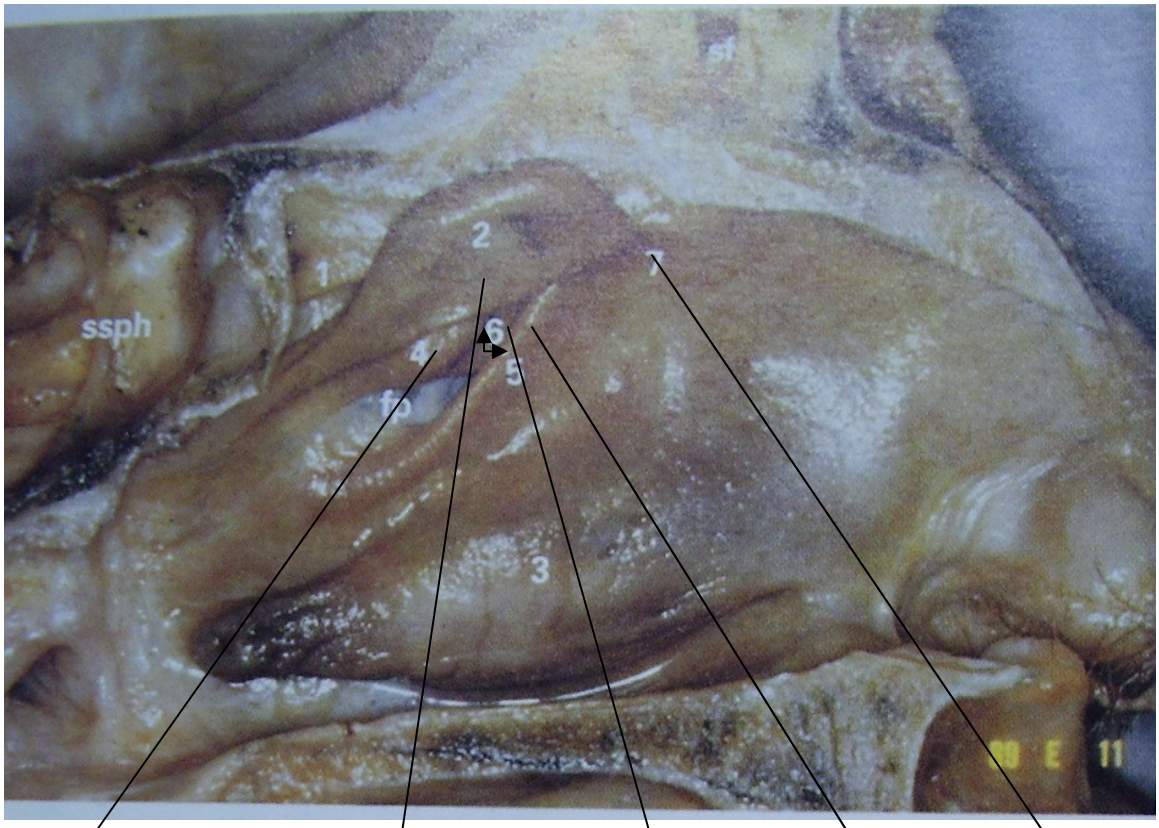


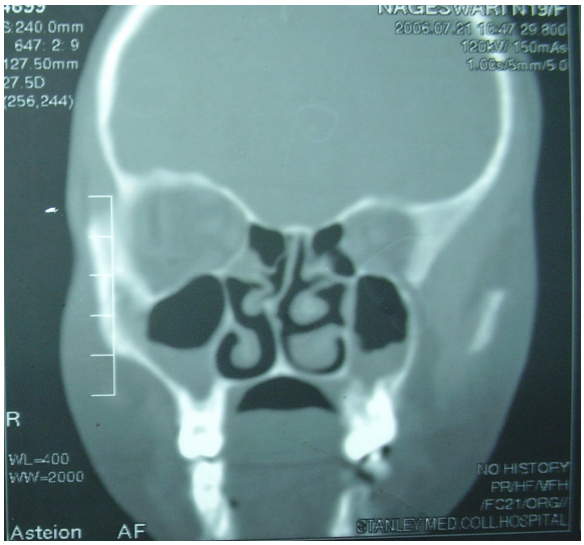
FIGURE 2.2 Schematic drawing of a beating cilium. Note how the cilium moves primarily in the sol phase (blue) and touches the gel phase (purple) only briefly during its fast "active beat," thus pushing the gel and the "blanket" in the direction of the active beat (to the right in this case). The slower "recovery beat" takes the cilium to the extreme left without touching the gel layer during this movement.

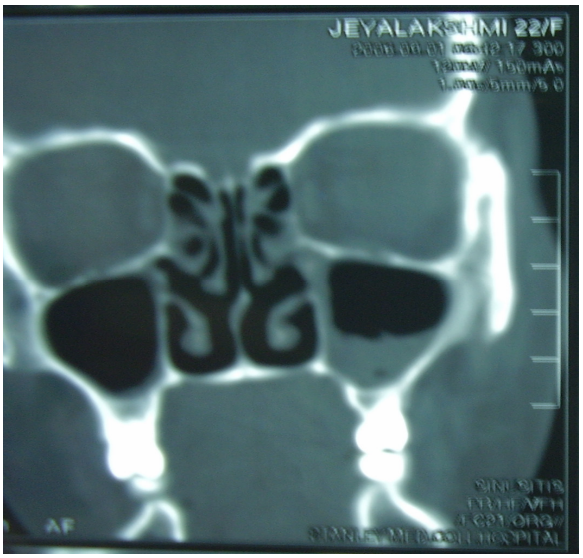
LATERAL WALL OF NOSE

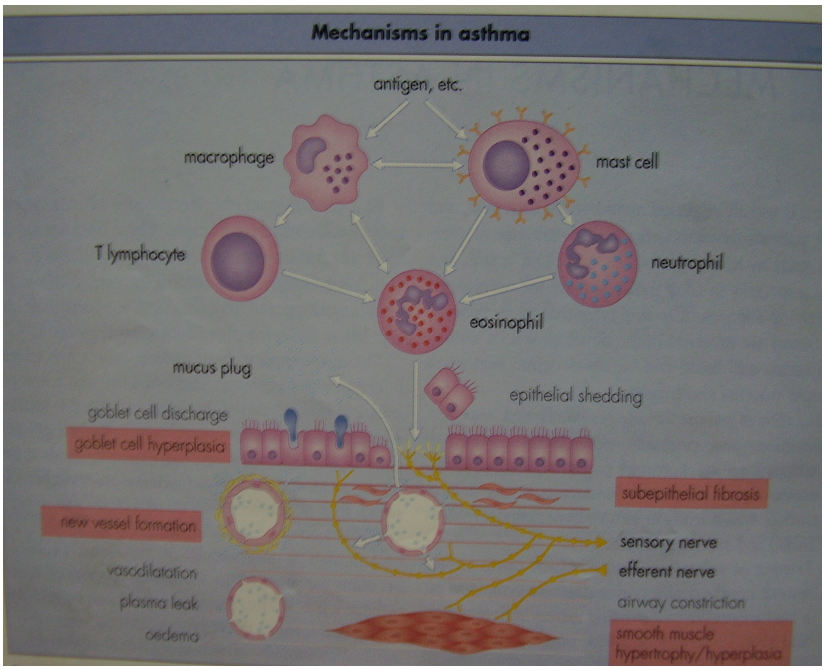


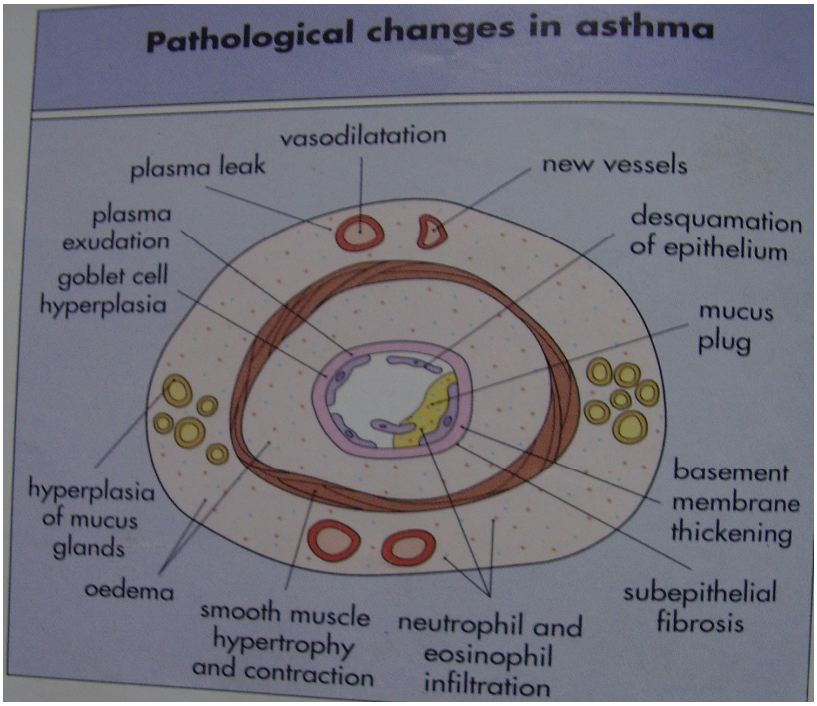
bulla **middle turbinate** **hiatus semilunaris** **uncinate** **aggernasi**

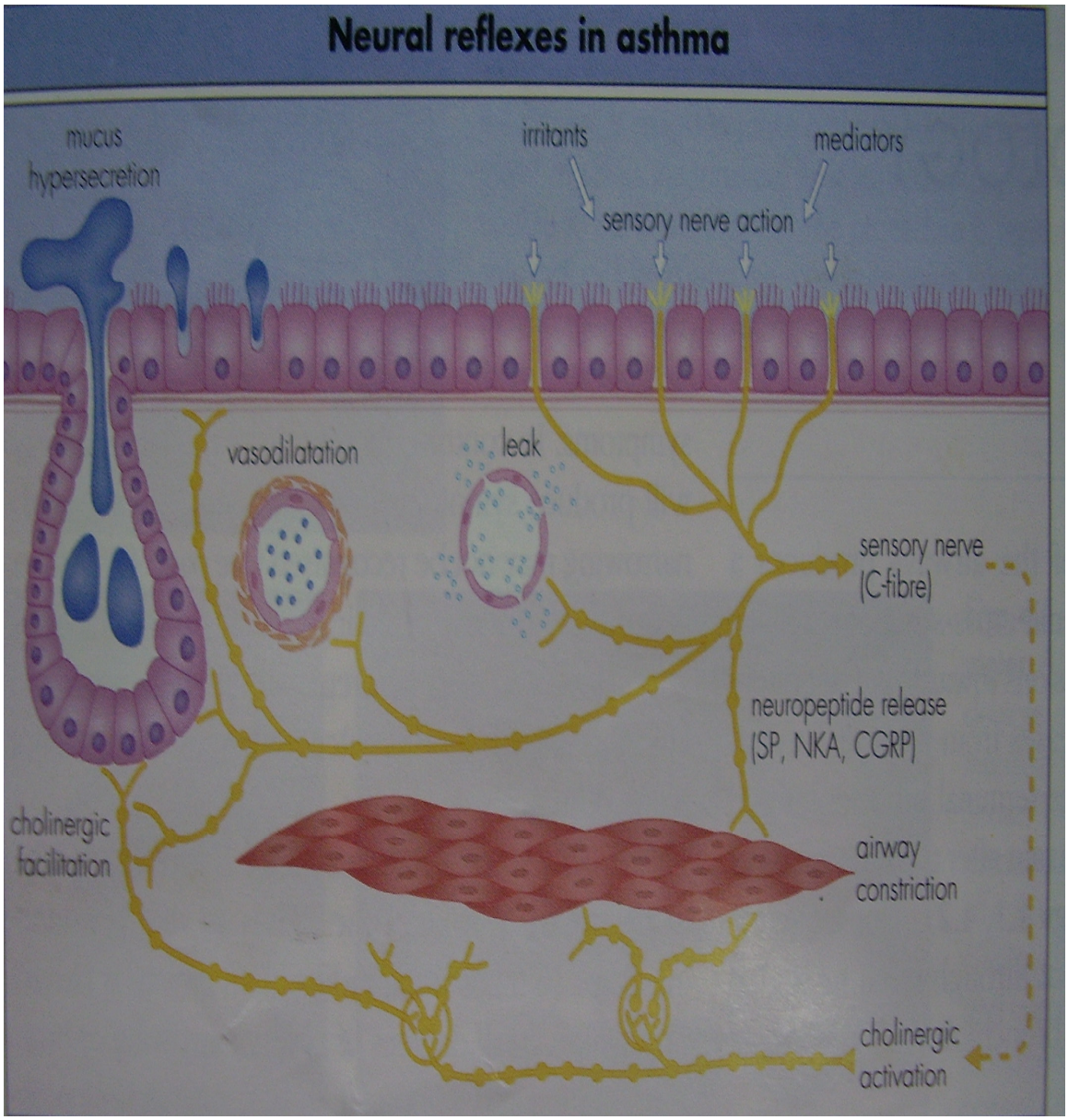
CT SCAN PARANASAL SINUSES CORONAL SECTIONS SHOWING EVIDENCE OF SINUSITIS

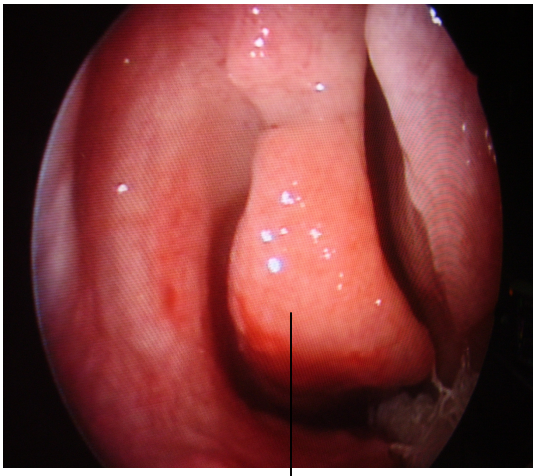




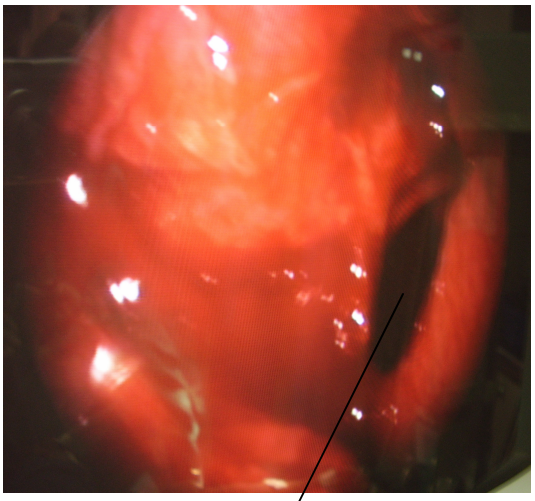

Mechanisms in asthma
antigen, etc.
macrophage
mast cell
T lymphocyte
neutrophil
eosinophil
mucus plug
epithelial shedding
goblet cell discharge
goblet cell hyperplasia
subepithelial fibrosis
new vessel formation
sensory nerve
efferent nerve
vasodilatation
plasma leak
oedema
airway constriction
smooth muscle
hypertrophy/hyperplasia


Pathological changes in asthma
vasodilatation
plasma leak
new vessels
plasma exudation
desquamation of epithelium
goblet cell hyperplasia
mucus plug
basement membrane thickening
hyperplasia of mucus glands
oedema
smooth muscle hypertrophy and contraction
neutrophil and eosinophil infiltration
subepithelial fibrosis


Neural reflexes in asthma
mucus hypersecretion
irritants
mediators
sensory nerve action
vasodilatation
leak
sensory nerve (C-fibre)
neuropeptide release (SP, NKA, CGRP)
cholinergic facilitation
airway constriction
cholinergic activation



bullous middle turbinate



wide middle meatal antrostomy